Endocranial Hyperostosis in Sangiran 2, Gibraltar 1, and Shanidar 5

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ABSTRACT Sangiran hominid 2 (S-2), Gibraltar hominid 1 (G-1), and Shanidar hominid 5 (SH-5) exhibit previously undescribed bilateral, paramedian hyperostosis of the endocranial frontal squama that spares the frontal crest, sagittal sinus, and ectocranial surface. The hyperostosis is localized to the frontal (usually the middle third) and parietal and is consistent with a diagnosis of hyperostosis calvariae interna (HCI), inclusive of hyperostosis frontalis interna. The hyperostosis in these specimens is compared to fossil hominids from Indonesia and Europe and to modern human cases of HCI. The three cases of HCI reported here document the existence and frequency of HCI in fossil hominids and push the antiquity of the disease back to nearly 1.5 millin years. The relatively great incidence of HCI in fossil hominids adds another confounding factor to the problematical issue of the taxonomic significance of cranial vault thickness. Am J Phys Anthropol 102:111–122.

Variation is crucial to natural selection. Interpretations of hominid phylogeny depend upon successfully differentiating between idiosyncratic and taxonomically salient variation (Schultz, 1963; Clark, 1978), whereas interpretations of individual behaviors, health, and history rely more heavily on idiosyncratic variation (Işçan and Kennedy, 1989). Pathological conditions in particular provide important information about an individual's quality of life as well as the history and evolution of disease (e.g., Richards and Antón, 1991; Rothschild et al., 1995). However, pathological conditions, unless grossly deforming, are often overlooked or dismissed in primary descriptions of fossil hominids as unimportant to the task of interpreting hominid phylogenies.

This paper addresses previously undescribed pathological endocranial hyperostosis in three fossil hominids: Sangiran hominid 2 (S-2), Gibraltar hominid 1 (G-1; Forbes Quarry 1), and Shanidar hominid 5 (SH-5). The pathological conditions are similar in appearance and may have a common origin in each hominid. Because the original SH-5 specimen could not be examined, this diagnosis must be considered tentative. The nature and distribution of the hyperostosis, its probable cause, differential diagnosis, and implications are discussed below with respect to other Indonesian and European fossil hominids and modern humans.

Pathology of endocranial hyperostosis

Endocranial hyperostosis is expansion in width of the endocranial surface of the vault involving either the inner table or the inner table and diploë; the outer table is unaffected. Expansion may occur throughout the vault and cranial base (hyperostosis cranii, HC) or may be restricted to portions of the vault (hyperostosis calvariae interna, HCI;

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Peruo, 1964). I limit subsequent discussion to HCI as neither S-2 nor G-1, which retain portions of the cranial base, exhibit cranial base hyperostosis.

Vault expansion in HCI may result from deposition of lamellar bone by the osteogenic layer of the dura onto the endocranial surface, although growth of the inner table/diploë directly via the endosteum is also possible (Moore, 1955). Subsequently, lamellar deposits are remodelled into diploë such that the thickness of the inner table is maintained or diminished and the diploë enlarged (Jaffe, 1972). Histological sections of hyperostotic areas exhibit "normal" bone components including Haversian systems and lamellar bone with no evidence of neoplastic cells. Thus HCI is dysplastic, not neoplastic, in origin (Perou, 1964).

The resulting endocranial surface is bumpy or nodular (Jaffe, 1972). In its earliest stages HCI consists of small plaques of bone deposited on the inner table. These plaques are not visible radiographically and are referred to as the "en-plaque" type (Fig. 1, grade O-I of Perou, 1964; Watrous et al., 1993, in preparation). These plaques enlarge to warty growths and massive or restricted nodular growths by additional dural deposition (Fig. 2; grade II and III of Perou, 1964; Jaffe, 1972). The edges of the growths become continuous with the cortical surface of the inner table as diploization occurs.

HCI has a distinctive pattern of distribution and progression that most frequently begins in and is limited to the frontal bone. The middle one-third of the frontal squama on either side of the midline is most severely predilected, although the orbital plate may also be involved (Perou, 1964; Jaffe, 1972). The hyperostosis, which is generally bilateral, may end abruptly at the middle meningeal artery grooves or may continue onto the parietal bones predilicting the edges of the sagittal sulcus (Moore, 1955; Perou, 1964; Jaffe, 1972). The sagittal sulcus itself is spared as are other, smaller blood channels (Jaffe, 1972). The temporal bones and occipital squama are least often affected and only after involvement of the frontal or parietal bones (Moore, 1955; Perou, 1964). Although hyperostosis of the frontal bone alone has been given its own name, hyperostosis frontalis interna (HFI; Moore, 1955), this hyperostosis often grades into the parietal bone and may be considered a subset of the wider HCI category (Perou, 1964). HCI is not associated with hyperostosis of other skeletal regions.

As will be discussed below, the particular pattern and distribution of hyperostosis differentiates HCI from other types of endocranial hypertrophy such as osteomas, HC, pregnancy osteophytes, Paget's disease, leontiasis ossea, and hyperostosis due to acromegaly, or Fröhlich's disease (Jelsma, 1959; Jaffe, 1972; Frame et al., 1987).

HCI appears to be an independent disease process although its cause remains obscure (Perou, 1964). Non-skeletal clinical signs often associated with HCI include headaches, and mental imbalance in elderly, obese females with hirsutism (Henschen, 1937; Moore, 1955; Perou, 1964; Jaffe, 1972). This association has led to the implication of various endocrine irregularities in the development of HCI and particularly HFI. Together, HFI and the aforementioned symptoms are known as Morgagni-Stewart-Morel-Moore syndrome (Morgagni, 1719; Stewart, 1928; Morel, 1929; Moore, 1955). However, HFI and HCI occur as often in isolation. In fact, older age is one of the few consistent indicators associated with HCI.

According to clinical studies, HCI is vastly more prevalent in females than in males (e.g., Henschen, 1937; Moore, 1955; Perou, 1964; Jaffe, 1972). In samples of otherwise normal individuals of all ages the general occurrence of HCI is between 3% and 15% (Moore, 1955; Jaffe, 1972). Commonly 12–16% of females are affected, whereas only 1–3% of males are affected (Moore, 1955; Jaffe, 1972). However, other age categories and independent studies reveal very different frequencies. In postmenopausal women the prevalence rate in some studies is as high as 40–60% (Henschen, 1937), whereas other studies of older women reveal prevalences of 15–25% (Moore, 1955). Despite the preponderance of HCI in older females, a number of young male cases also exist, especially where HCI is a secondary character to an overtly genetic disorder such as dystrophia myotonica (Caughey, 1952). Archaeological cases indicate that the preva-

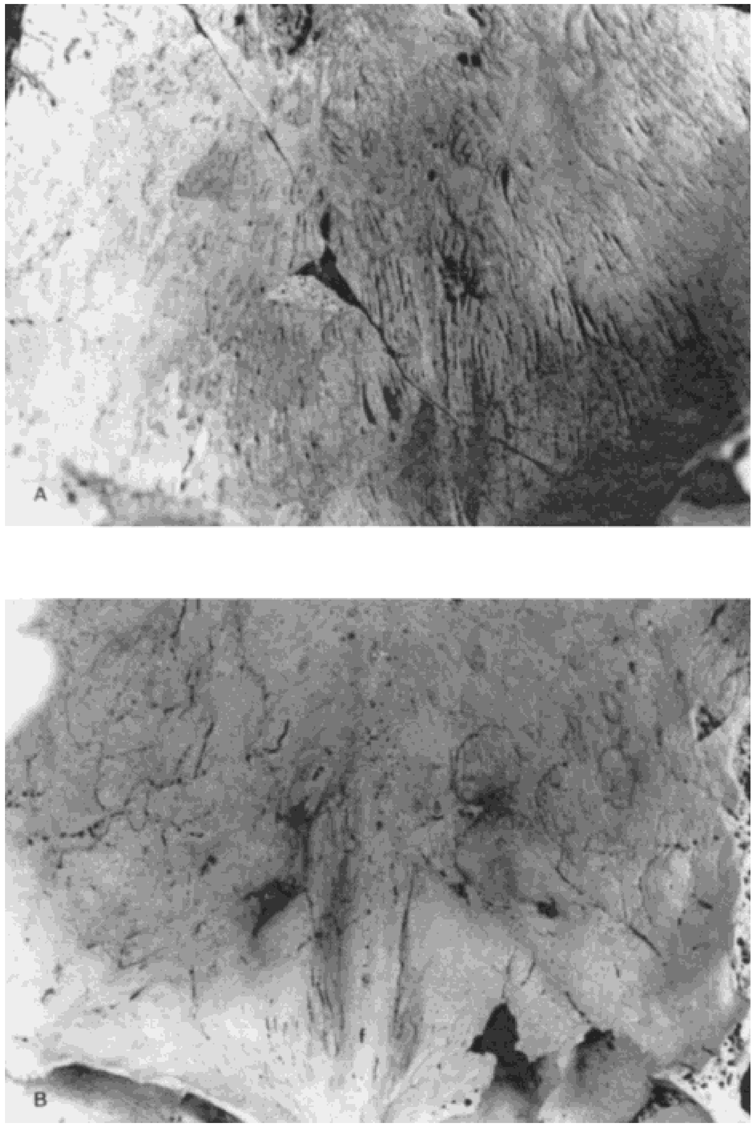

Fig. 1. Posterior view of the endocranial frontal squama of early "en-plaque" stages of HCI in modern humans from Giza, Egypt. Superior is toward the top of the page. f = frontal crest. **A:** HMA 12-5183. Note numerous small plaques deposited on the endocranial surface. **B:** HMA 12-5148. Grade I. Note larger sized plaques, some with edges blending into endocranial surface. Note that the sagittal sulcus is spared.

lence of male HCI is higher than clinically appreciated (Watrous et al., 1993).

Paleopathology of HCI

Only 13 cases of HCI are reported in ancient human remains (Table 1). Of these, the earliest and most numerous cases are from ancient Egypt and Nubia. This preponderance may reflect the relative longevity of these populations as well as the availability of relatively large samples. The most ancient cases previously reported are three individuals from the Fourth to Fifth dynasties at Giza, Egypt (ca. 2630–2350 BC; Wa-

Fig. 2. Posterior view of the endocranial frontal squama of later stages of HCI in modern human cadaver samples. Superior is toward the top of the page. Note that the midline and endocranial vessel channels are spared. **A:** Grade II smooth type. The entire frontal squama is expanded with nodules incorporated into the endocranial surface. **B:** Grade III. Note the hyperexpansion and billowy appearance of the frontal squama and the further superior extension of the hyperostosis onto the frontal and parietal.

trous et al., 1993). Remarkably, two of the three are males from the same Mastaba group (2300 BC). If the Mastaba contained closely related individuals, these cases may lend support to the genetic underpinning of male HCI. These cases and an additional case from Naga-ed-Deir, Egypt (ca. 2200–1800 BC) await fuller reports by Watrous et al. (1993, in preparation). Two Meroitic period cases (ca. AD 300) from Nubia have been reported by Armelagos and Chrisman (1988) and Nielsen (1970). These cases appear in females of 40-years and "middle-age," respectively. Non-African cases include a 60-year-old female from the Isola Sacra necropolis, Italy (first to third centuries AD; Sperdutti and Manzi, 1990), a 30–40-year-old female from Sarre, Kent (sixth century AD, Anderson, 1993), and an "older" female from 10th century Norway (Henschen, 1937).

Other extensive surveys of archaeological skeletal material have failed to produce further cases of HC/HFI (Moore, 1955). In an extensive survey of HC, Moore (1955) could not identify a single case in the archaeological skeletal collections of the Smithsonian Institution, American Museum of Natural History, or Field Museum of Natural History. However, it is not clear whether he examined entire collections or a subset of specimens from each. In addition, Moore failed to observe a single case of endocranial hyperostosis in 162 Mound-Builder skulls of unspecified origin. However, he documented 99 cases among 1,478 skulls in the Terry collection.

HCI has not been documented in fossil hominids. However, Moore (1955) believed that the endocasts of Gibraltar (presumably the adult), Mousterian (Le Moustier?), and a *Pithecanthropus* (locality/number unknown) housed at the Field Museum of Natural History exhibited imprints similar to those seen on the frontal lobe of the brain in HFI. Moore never examined casts or originals of the endocranial frontal bone of these hominids. Without prior knowledge of Moore's suggestions, and in the course of research for other purposes, I noted endocranial hyperostosis in the original Sangiran 2 and Gibraltar 1 hominids and casts of Shanidar 5. The nature and distribution of these hyperostoses is described below.

MATERIALS AND METHODS

Endocranial surfaces of Indonesian *Homo erectus*, European Neandertals, and modern *Homo sapiens sapiens* were examined for bony hyperostosis (Table 2). The non-fossil modern humans included archaeological crania from Egypt and dissecting room specimens (n = 28) examined as part of a previ-

TABLE 1. Distribution of archaeological cases of hyperostosis calvariae internal hyperostosis frontalis interna (HCI/HFI)

Site	Date	Specimen	Citation
Giza, Egypt	2630–2350 BC	2 males (35 and 45 years) 1 female (30 years)	Watrous et al., 1993
Naga-ed-Deir, Egypt	220–180 BC	1 female (advanced age)	Watrous et al., 1993
Sudanese Nubia	AD 300	1 female (40 years)	Armelagos and Chrisman, 1988
Sudanese Nubia	0–AD 350	1 female (40 years)	Neilsen, 1970
Isola Sacra, Italy	1st–3rd centuries AD	1 female (50–60 years)	Sperdutti and Manzi, 1990
Sarre, Kent, UK	6th century AD	1 female (30–40 years)	Anderson, 1993
Stuttgart, Germany	5–7th centuries AD	1 female (late adult)	Hahn and Czarnetski, 1980
Oseberg, Norway	10 century AD	1 female (older)	Henschen, 1937
Szczecin, Poland	14–15th centuries AD	1 female (50–60 years)	Gladykowska-Rzeczycka, 1988
Crailsheim, Germany	18th century AD	1 female (72 years)	Hahn and Czarnetski, 1980
Philadelphia, USA	19th century AD	1 female (27 years)	Angel et al., 1987

ous study of the relative distribution and etiological implications of HFI (Watrous et al., 1993, in preparation). Skull caps from some of the affected cadavers are housed in the Atkinson collection, University of the Pacific, School of Dentistry (UOP, SRA). Modern human cases are presented here as a basis for comparison with the fossil hominids. More detailed discussion of the modern human cases is provided by Watrous et al. (1993, in preparation).

Evidence of endocranial hyperostosis was sought in each hominid by direct endocranial observation. Hyperostosis was considered present if any of the following distinct changes of the endocranial surface were observed: 1) slight but clearly detectable deposits of bone on the inner table of the vault with edges distinct from the endocranial surface (Fig. 1A; "en-plaque" type, not visible radiographically), 2) multiple distinct bony nodules with some edges continuous with the inner table (Fig. 1B; grade I of Perou, 1964), 3) unusual expansion of diploic or cortical origin of a localized region of the endocranial surface presenting either a smooth or warty surface (Fig. 2; grades II and III of Perou, 1964). In the absence of these changes to the surface anatomy of the inner table, absolute skull thickness was not considered indicative of pathological hyperostosis.

TABLE 2. Samples and sources

Sample	Source
H. erectus	
Trinil	RM
Sangiran, 2, 3, 4	SMF
Sangiran 12, 17	GRDC
Ngandong 2	GMU
Ngandong 5, 9, 10—casts	UF/SMF
Neandertals	
Gibraltar 1, 2	BMNH
Tabun C1	BMNH
Shanidar V-cast	UCB/MH
La Ferrassie 1, 2, 3	MH
La Quina 5	MH
Pech de L'Aze	MH
Fossil *H. sapiens*	
La Madeleine 1	MH
Cro-Magnon 1, 2, 3, 4	MH
Recent *H. sapiens*	
Giza, Egypt	HMA
Naga-ed-Deir, Egypt	HMA
U.S. cadavers	UOP/UCB

Locations of collections are abbreviated as in the text.

The type and extent of hyperostosis was recorded for each individual. The distribution and pattern of hyperostosis in each of the fossil hominids is described in detail below.

RESULTS

Descriptions of pathological hyperostoses

Sangiran 2. Sangiran 2 was recovered from the lower part of the Bapang (Kabuh) formation of the Sangiran dome, Java Indonesia in 1937 (von Koenigswald, 1940). Based on paleomagnetic studies and fission track dates from the middle tuff and tektite layer above and Tuff 10 of the Sangiran (Pucangan) formation below, the lower Bapang formation is suggested to date between 0.78 and 1.16 mya (Suzuki, et al., 1985; Itihara et al., 1994). However, $^{40}Ar/^{39}Ar$ dating suggests the upper part of the Sangiran formation dates to 1.66 mya (Swisher et al., 1994), and $^{40}Ar/^{39}Ar$ work and paleomagnetics in progress suggest the lower tuff of the Bapang formation may be as old as 1.5 mya (Swisher, 1966, personal communication).

Sangiran 2 is an adult *Homo erectus* calvaria lacking much of the cranial base and the entire face. Because its cranial sutures are fused endo- and ectocranially, S-2 is considered to have been older at the time of its death than other Sangiran hominids except Sangiran 4 (von Koenigswald, 1940; Weidenreich, 1941; Oakley et al., 1975). The left glenoid fossa also exhibits use or age-related degenerative changes. Because of its small size, S-2 is usually considered to be female (von Koenigswald, 1940; Oakley et al., 1975).

Neither von Koenigswald's (1940) original description nor subsequent descriptions of S-2 (e.g., Weidenreich, 1943; Rightmire, 1993) record the presence of endocranial nodules. This is understandable given that these works principally examine the phylogenetic position of the Sangiran specimen. Except for Moore's (1955) reference to a *Pithecanthropus* endocast, no mention has been made of HFI/HCI in *Homo erectus.*

Sangiran 2 exhibits mild hyperostosis of the "en-plaque" type on the frontal and parietal bones (Fig. 3). The frontal bone is most severely affected. The largest, most defined plaques sit on either side of the frontal crest, near its distal end (Fig. 3). The smaller plaque is on the right side (6.6 × 5.0 mm), the larger on the left (15.0 × 6.0 mm). Anterior to the larger plaque are plaques associated with the edges of two linear, but converging, grooves that may have contained blood vessels during life. It is impossible to know if these linear structures were bilateral as the right side of the frontal bone is absent in this region. Posterior to the frontal crest, distributed randomly about the endocranial frontal squama, are numerous, small plaques. Hyperostosis on the parietal bones is limited to small beads of bone along the lateral edges of the sagittal sulcus and one bone ridge that crosses the sulcus anteriorly. The bone beads begin approximately 25 mm posterior of bregma and extend posteriorly approximately 45 mm along the sagittal sulcus. The hyperostosis of S-2 is most similar to that in modern humans from Giza (Fig. 1B).

Shanidar 5. Shanidar 5 was recovered in situ in the upper level of the Middle Paleolithic layer (layer D) of Shanidar Cave, Zagros Mountains, Iraq in 1960 (Trinkaus, 1983). Upper layer D has an associated radiocarbon date of 46,900 ± 1,500 years BP (Trinkaus, 1978).

Shanidar 5 is an adult Neandertal partial cranium preserving the face and left side of the cranial vault (Trinkaus, 1978, 1983). Sutural fusion and dental wear suggest Shanidar 5 is the oldest of the Shanidar adults (Trinkaus, 1978, 1983). Size and robusticity suggest SH-5 is a male (Trinkaus, 1978, 1983). In his original descriptions and subsequent reports of pathological conditions, Trinkaus (1978, 1983) does not discuss the endocranial plaques of SH-5.

It was not possible to examine the original SH-5 specimen, but high-quality casts of both endo- and ectocranial surfaces were examined at the Musée de L'homme, Paris (MH), and the Laboratory for Human Evolutionary Studies, University of California, Berkeley (UCB). The casts are considered adequate for this description and interpretation since high-quality casts of both S-2 and G-1 faithfully reproduce their endocranial hyperostoses.

The SH-5 frontal bone presents paramedian endocranial hyperostosis of the "en-plaque" type (Fig. 4). The plaques are located on either side of the frontal crest and the frontal part of the sagittal sinus. The plaques are larger than those of S-2 but are

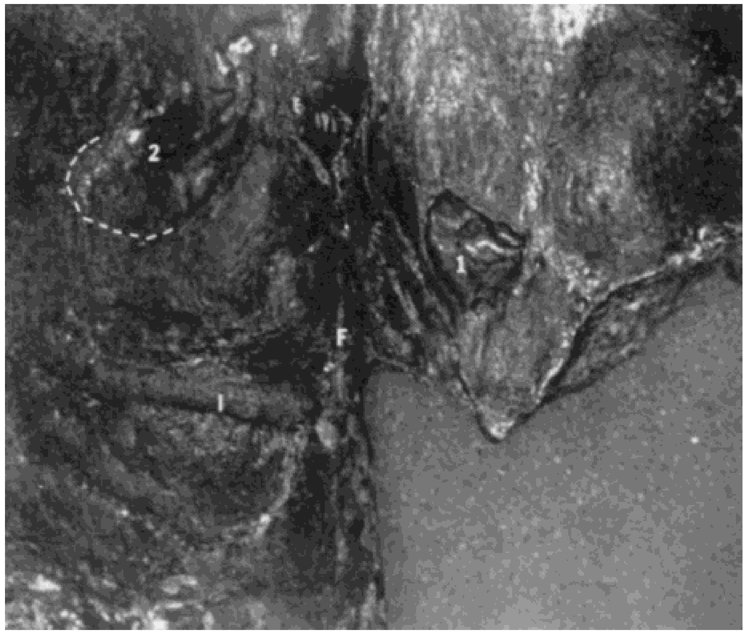

Fig. 3. Posterior view of the endocranial frontal squama of Sangiran 2 (the right side is partially absent lateral to the frontal crest). Grade I. F = frontal crest. Note large plaque (1) on right side. Note converging linear grooves on inferior left side and superior to these a large plaque (2).

restricted to the frontal squama. The left side of the frontal bone is slightly more hyperostotic than the right (Fig. 4). The SH-5 case is most similar to the hyperostosis seen in individuals from Giza (Fig. 1B).

Gibraltar 1. The Gibraltar 1 hominid was discovered in Forbes' Quarry at the north base of the Rock of Gibraltar in 1848 but did not receive scientific attention until the 1860s (Hrdlička, 1930). There are, apparently, no precise records of how and where the specimen was found, although matrix on the cranium was associated later with descriptions of the strata. These strata have been dated to 45,000–70,000 BP based on interpretation of the deposits as "early Würm" (Oakley, 1964:150). However, this relative correlation is not convincing (Oakley, 1964:147), leaving both the stratigraphic position of the fossil within the quarry and the age of the quarry deposits unconfirmed.

G-1 is an adult Neandertal cranium lacking portions of the vault including the left parietal and temporal bones, portions of the left frontal and occipital bones, and the right parietal bone adjacent to the sagittal suture. The sagittal suture is not preserved. The right coronal suture is fused both endo- and ectocranially. The right lambdoidal, parietomastoid, and squamosal sutures are fused endocranially, but sutural traces are visible ectocranially. The right occipitomastoid is patent endo- and ectocranially. The preserved teeth are well worn. For these reasons, Broca (1869) considers G-1 older than 60 years of age in his original description. Although this age may be an overestimate, G-1 was clearly an older adult at the time of death. Because of its relatively small size, G-1 is usually considered to be female (Sera, 1910; Oakley et al., 1971).

In his original description of the Gibraltar cranium, Broca (1869) noted the endocranial nodules on the frontal. However, he suggested that the ectocranial surface of the G-1 frontal exhibited signs of a healed trauma and that the endocranial nodules were the result of the healing process. There is, however, no evidence of ectocranial trauma and endocranial hyperostosis, diploization, and nodular growths as seen in

Fig. 4. Posterior view of a cast of the endocranial frontal squama of Shanidar 5. Grade I. f = frontal crest, s = sagittal sulcus. Entire area of hyperostosis is delimited by the dashed line. Note small plaque to right of sagittal sulcus and area of plaques demarcated by < to left and superior.

G-1 are not typical of trauma induced healing in the cranium in any event (Ortner and Putschar, 1985).

G-1 exhibits the most severe hyperostosis of the three cases described here. Hyperostosis is of the warty/nodular type (grade II) and is apparently restricted to the frontal bone (Fig. 5). However, postmortem endocranial damage to the parietal and occipital may obscure this observation. Because the condition in G-1 is further advanced than in S-2, the edges of the nodules appear continuous with the endocranial surface rather than like plaques applied to the surface. A few bony plaques also remain in G-1 (Fig. 5). As revealed by postmortem fractures, the frontal exhibits an expanded diploë and a thin endocranial table.

The hyperostotic region circumscribes a triangular area on the lower, central portion of the frontal squama (Fig. 5). The apex of the triangle is located at the inferior midline of the frontal crest and its base is midway between the frontal crest and bregma. The hyperostotic region is thus wider superiorly than inferiorly. The base of this triangle is approximately 50 mm. The hyperostosis is most pronounced immediately adjacent to the frontal crest and decreases laterally; the actual midline and frontal crest are not affected. The right side has larger nodules than the left. The hyperostosis appears to end shortly after bifurcation of the frontal crest into the superior sagittal sulcus, although postmortem damage may obscure the presence of hyperostosis on the parietal and occipital. G-1 presents grade II hyperostosis as defined by Perou (1964) and most closely resembles modern human cadaver cases (Fig. 2) and the Nubian cases (Nielson, 1970, plate 19; Armelagos and Chrisman, 1988, their Fig. 1).

DISCUSSION

The fossil hominids discussed in this study exhibit bilateral, paramedian hyperostosis predilecting the endocranial frontal squama and sparing the frontal crest, sagittal sinus,

Fig. 5. Posterior view of the endocranial frontal squama of Gibraltar 1. Grade II. f = frontal crest, s = sagittal sulcus, deepened by hyperostosis. Note plaques (e.g., p) which are partially incorporated into the endocranial surface. Overtly hyperostotic region is demarcated by the dashed line. Note thickness of vault wall at left superior edge of figure.

and ectocranial surface. The hyperostosis is localized to the middle third of the frontal bone and edges of the sagittal sulcus of the parietal bones. The cranial base and face are not affected in the fossil hominids that preserve these regions. The appearance and distribution of the hyperostoses is consistent with a diagnosis of hyperostosis calvariae interna (HCI), inclusive of hyperostosis frontalis interna.

HCI can be differentiated from other sources of cranial hypertrophy including HC, Fröhlich's syndrome, Paget's disease, leontiasis ossea, acromegaly, osteomas, and pregnancy osteophytes by the appearance and distribution of the hyperostosis. HCI is differentiated from HC, Fröhlich's syndrome, and Paget's disease by lack of cranial base involvement (Moore, 1955; Perou, 1964; Frame et al., 1987). Paget's disease also involves the ectocranium and results in partial or complete loss of the diploë (Perou, 1964; Frame et al., 1987). Leontiasis ossea involves the facial skeleton (Perou, 1964). Cranial expansion due to acromegaly involves both endo- and ectocranial surfaces as well as enlargement of the frontal sinus and mandible (Perou, 1964; Jaffe, 1972). The hominids do not exhibit expanded cranial sinuses, or involvement of the cranial base, ectocranial vault, or face, although mandibles are not preserved. Osteomas tend to be unilateral, circumscribed, and single (Jelsma, 1959). Pregnancy osteophytes (PO) are small, predilect the frontal and parietal bones, and in particular the groove for the meningeal artery and the sagittal sinus but also appear ectocranially on the vault and face (Perou, 1964). PO are smaller in size than the Gibraltar nodules. The appearance of PO is more consistent with the S-2 and SH-5 cases, although SH-5 is probably male. Additionally, PO tend to be less diffuse on the frontal bones, more prevalent ectocranially, and to predilect rather than spare the venous sinuses. Thus none of these alternate sources of cranial hypertrophy are con-

sistent with the signs exhibited by the fossil hominids.

The three cases of HCI reported here present a significant addition to the small number (n = 13) of previously reported archaeological cases and to the number of presumed male cases. The fossil cases support the association between older age and HCI. However, in light of the clinical preponderance of female HCI, it is remarkable that one of three fossil cases is a presumed male. This case, in conjunction with those of Watrous et al. (1993), suggests HCI is a poor indicator of sex in skeletal remains.

Likewise, both studies suggest that male cases are more frequent than clinically appreciated, or were more frequent in past populations. Differentiation between these possibilities is difficult due to small skeletal sample sizes and the different diagnostic tools used in clinical and dry-bone studies. Dry-bone studies rely on direct examination of the endocranial surface, whereas clinical studies rely on radiographs. Early cases of HCI are radiographically invisible thus confounding comparisons between clinical and dry-bone frequencies; of the cases in this study only the Gibraltar 1 case would appear on a radiograph. In addition, the inability to recognize early cases of HCI radiographically likely produces a false demographic profile that obscures the etiology of the disease. Controlled examinations of age and sex distributions of HCI in archaeological and modern skeletal samples, controlling for variables such as environment and genetics, would begin to address this problem.

Additionally, clinical recognition is often accidental because HCI is generally asymptomatic until very advanced stages when the hyperostosis may impinge on neural tissue (Moore, 1955). Thus clinical correlation between HCI and older, obese women with hirsutism and mental impairment probably reflects only a subset of the true cases. The assumption that endocrine irregularities are the ultimate source of the HCI, as they are of the obesity and hirsutism, may thus be erroneous. HCI may prove to be the result of a more generalized aging process, as suggested by the relatively consistent association of older age with both clinical and archaeological cases HCI (e.g., Jaffe, 1972 and Table 1). Again, controlled examinations of age and sex distributions of HCI in modern skeletal and cadaver samples could address the process, pattern, and distribution of HCI. Even the question of whether the original source of the hyperostosis is dural or endosteal might be resolved through evaluation of cadaver samples with dural tissue still in place.

In addition to their implications for clinical interpretations, the relatively great prevalence of HCI in fossil hominids adds another confounding factor to the interpretation of the taxonomic significance of cranial vault thickness (e.g., Hublin, 1991; Brown, 1994). The cases of S-2 and SH-5 are probably not advanced enough to influence thickness measurements except when an endocranial plaque is directly included in the measurement. However, the G-1 individual exhibits morphological evidence of diploic expansion throughout the frontal bone (Fig. 5). This expansion is more advanced than in S-2 or SH-5 and certainly affects measurements of cranial thickness throughout the frontal bone. Because the etiology of HCI is nebulous, it is unknown whether cranial thickness in other parts of the vault is entirely unaffected. Given the variety of causes, including disease, that result in increases in absolute vault thickness, "thick vault" cannot be considered a good taxonomic character.

The fossil hominid cases push the antiquity of HCI back to perhaps 1.5 million years ago and into two fossil groups of the genus *Homo.* Sangiran 2 is the most ancient case of HCI yet reported and is the only Indonesian hominid to exhibit endocranial hyperostosis. Even the Trinil specimen, which exhibits a similar degree of sutural fusion, is entirely free of hyperostosis. The stratigraphically older Sangiran 4 specimen shows no evidence of HCI, although S-4 does not have a frontal bone. Older cases may exist in African hominids. G-1 and SH-5 are the only Neanderthals examined that exhibit endocranial hyperostosis. However, because original anatomical descriptions cannot always be relied upon to reflect the presence of HCI, it is possible that Neanderthals not examined here may exhibit HCI.

The presence of HCI in older individuals from two fossil hominid groups and modern humans suggests that HCI results from perturbation of a cranial growth/aging process common to members of the genus *Homo*.

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